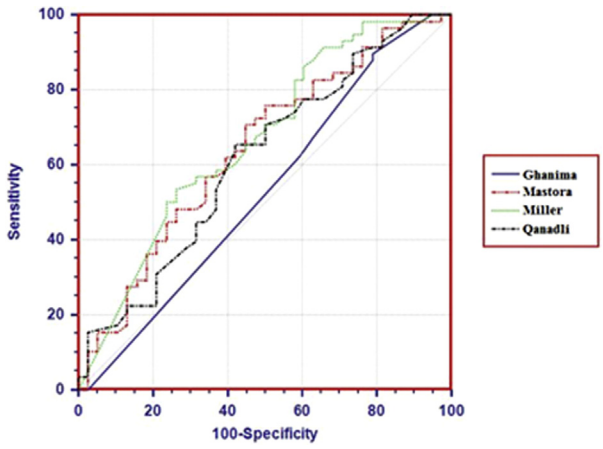

Table 1. The demographic- clinical characteristics and CT- echocardiographic parameters of patients with and without RVD

Variables	Patients without RVD (n=50) Mean ±SD/Median(IQR)	Patients with RVD (n=70)Mean ±SD/Median(IQR)	P value
Age (years)	63.8 ± 17.8	65.8 ± 12.6	0.50
Gender n (F/M)	30/20	34/36	0.14
Hypertension n (%)	16 (32.0)	30 (42.9)	0.23
Systolic BP (mmHg)	110.3 ± 17.3	111.3 ± 24.2	0.81
Diastolic BP (mmHg)	70.5± 11.8	69.8 ±13.1	0.77
Pulse (beats per minute)	94.8 ± 15.5	91.0 ± 15.3	0.24
Troponin (ng/mL)	0.65 ± 1.8	2.21± 8.2	0.74
Geneva Score	7.8 ± 3.7	7.5 ± 3.8	0.69
Wells Score	5.6 ± 1.9	5.5 ± 1.9	0.81
Intubation	0 (0)	2 (2.9)	0.15
Non-invasive mechanic ventilation	1 (2.0)	5 (7.1)	0.20
in-hospital mortality	2 (4.0)	4 (5.7)	0.72
Combined end- point	3 (6.0)	11 (15.7)	0.04
RV/LV Ratio	1.12 ± 0.22	1.25 ± 0.20	0.002
SVC axial diameter (mm)	20.2 ± 3.6	22.5 ± 3.4	0.001
PA axial diameter (mm)	29.7 ± 4.2	33.3 ± 5.5	<0.001
Ghanima	3.4 ± 1.0	3.5 ± 0.7	0.44
Miller	65.1 ± 29.9	81.2 ± 23.5	0.004
Qanadli	44.3 ± 22.8	53.5 ± 20.7	0.045
Mastora	29.9 ± 17.3	37.6 ± 16.3	0.029
LV-EDD (cm)	4.4±0.4	4.4±0.5	0.55
LV-ESD (cm)	2.6±0.5	2.7±0.5	0.76
EF (%)	59.7±5.7	59.6±3.7	0.91
PAP systolic (mmHg)	39.7±13.7	58.6±14.7	<0.001
RV Sm cm/s	13.93 ± 2.65	11.80±3.69	0.07

BP: Blood Pressure, EDD: End diastolic diameter, ESD: End Systolic diameter, EF:Ejection fraction, IQR: Inter quartile range, LV:Left Ventricule, PA:Pulmonary Artery, PAP: Pulmonary artery pressure, RV: Right Ventricule, RV Sm: Right ventricular myocardial systolic velocity, SVC: Superior Vena Cava

Epidemiology

Monday, October 28, 2013, 14:00 PM–15:15 PM
Hall: SARAJEVO

Abstract nos: 117-122

OP-117

Gene-Gene Interaction between APOA4 and FTO for Obesity in TARF Study

Filiz Geyik[1], Neslihan Çoban[1], Berna Yüzbaşıoğulları[1], Altan Onat[2], Vedat Sansoy[3], Can Günay[4], Nihan Erginel Ünaltuna[1]
[1]Department of Genetics, Institute for Experimental Medicine, Istanbul University, Istanbul, [2]Emeritus Professor, Department of Cardiology, Cerrahpasa Medical Faculty, Istanbul University, Istanbul, [3]Department of Cardiology, Institute of Cardiology, Istanbul University, Istanbul, [4]Department of Public Health, Cerrahpaşa Medical Faculty, Istanbul University, Istanbul

Aim: Obesity, a major cluster of risk factors for cardiovascular diseases, shows increasing prevalence worldwide. The risk of obesity is determined not only by specific genotypes but also by significant gene-gene interactions. Several studies have established associations of both apolipoprotein A4 (APOA4) gene variants and fat mass and an obesity associated (FTO) gene variants with blood lipid levels and obesity. Our aim was to explore the synergistic contribution of the two polymorphisms: T347S of the APOA4 gene and rs1421085 of the FTO gene to obesity risk in a Turkish adult population.
Methods: We investigated a possible interaction between these two genes on the risk for the obesity, using data from the TARF study (1512 men and women aged 50.06±12.01 years). Genotyping was achieved by high throughput systems as Real-Time PCR LC480 and ABI-7900HT. Statistical analysis was performed by conditional multiple regression models.
Results: APOA4 T347S and FTO rs1421085 polymorphism combinations showed a nominal gene-gene interaction (p values 0.014 and 0.030 respectively) on obesity. Logistic regression analyses of T347S ve rs1421085 polymorphisms, after adjustment for age and gender these interactions were also showed (p for interaction =0.007 and =0.027, respectively).
Conclusion: A synergistic effect between polymorphism T347S of the APOA4 gene and rs1421085 of the FTO gene for obesity risk was found in Turkish adult population.

OP-118

Genetic Variation in LPA Gene Predicts Plasma Lipoprotein(a) Level and Type 2 Diabetes Risk

Neslihan Çoban[1], Altan Onat[2], Günay Can[3], Nihan Erginel Ünaltuna[1]
[1]Department of Genetics, Institute for Experimental Medical Research, Istanbul University, Istanbul, [2]Department of Cardiology, Cerrahpasa Medical Faculty, Istanbul University, Istanbul, [3]Department of Public Health, Cerrahpasa Medical Faculty, Istanbul University, Istanbul

Aim: Recent studies found that Lipoprotein(a) [Lp (a)] levels were lower in patients with type 2 diabetes than the non-diabetics. Little is known about the genetic determinants for Lp(a) levels in diabetic patients, and it remains unclear whether elevated Lp(a) levels may causally affect cardiovascular disease risk in the diabetic patients. LPA encodes apolipoprotein(a), and rs10455872 polymorphism in the LPA locus is associated with Lp(a) levels and cardiovascular risk factors. In this study, therefore, we aimed to investigate the effects of the rs10455872 polymorphism in the LPA gene locus on Lp(a) levels and risk factors for type 2 diabetes in the Turkish population.
Method: We examined one single-nucleotide polymorphism (SNP) in LPA gene in the Turkish Adult Risk Factor (TARF) Study DNA bank which has been established between 2004-2010 years. The sample was comprised of 2252 Turkish adults. Genotyping was performed by high throughput system, Real-Time PCR LC480 device. The association between biochemical, clinical parameters and the polymorphism have been analyzed using SPSS software. For continuous variables, ANOVA T-test was used, whereas X^2 analysis was performed for categoricals.
Results: In this study, the first one to examine the rs10455872 polymorphism of the LPA gene in the Turkish population. The distribution of the LPA rs10455872 polymorphism in this adult population was 97% (n=2185), 3% (n=66) and 0% (n=1) for the AA, AG and GG genotypes, respectively. The rs10455872 in LPA gene locus was most strongly associated with higher Lp(a) levels ($p<0.0001$) in Turkish adults. In contrast, the LPA rs10455872 AG genotype was correlated lower HOMA index and insulin levels in Turkish adults ($p<0.01$). However, no significant relationship was found for the LPA rs10455872 polymorphism with type 2 diabetes in men and women.
Conclusion: The LPA rs10455872 AG genotype appears to be a risk factor against coronary heart disease by increasing the Lp(a). In contrast, AG genotype of the LPA gene independently confers protectiveness for type 2 diabetes by decreasing the HOMA index and insulin levels in Turkish adults.

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